Original Article

Prevalence of Canine Infectious Endocarditis and Possible Association with *Bartonella* spp. in Bangkok, Thailand

Sirilak D. Surachetpong[1*] Sukullaya Assarasakorn[1] Anudep Rungsipipat[2] A. Valeria Scorza[3] Melissa M. Brewer[3] Kenneth W. Simpson[4] Michael R. Lappin[3]

Abstract

Infectious endocarditis (IE) is a heart valve or endocardial disease. *Bartonella* spp. are considered one of the causes of IE. The objective was to study the prevalence of canine IE in Bangkok, Thailand with an emphasis on *Bartonella* spp. infections. A review of the reports between January 1999 to December 2009 of 3,545 necropsied dogs was performed. Cardiac tissue blocks from 11 dogs were studied for the presence of eubacterial DNA and *Bartonella* spp. DNA by fluorescence *in situ* hybridization (FISH) and for *Bartonella* spp. DNA by conventional polymerase chain reaction (PCR).The prevalence of canine IE was 0.65%. The cardiac tissues from 2 of 11 dogs were positive for eubacteria DNA by FISH. None of the dogs was positive for *Bartonella* spp. by FISH or conventional PCR. The prevalence of canine IE was low in this population and *Bartonella* spp. DNA was not detected in any dog tested with these techniques.

Keywords: *Bartonella* spp., dogs, endocarditis, heart, Thailand, valve

[1] *Department of Veterinary Medicine, Faculty of Veterinary Science, Chulalongkorn University, Bangkok 10330, Thailand*
[2] *Department of Veterinary Pathology, Faculty of Veterinary Science, Chulalongkorn University, Bangkok 10330, Thailand*
[3] *Department of Clinical Sciences, Colorado State University, Fort Collins, CO 80523, USA*
[4] *Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, Ithaca, NY 14853, USA*
[*] ***Correspondence:*** *sirilakd27@gmail.com*

Introduction

Infectious endocarditis (IE) is a heart valve or an endomyocardial disease caused by bacterial infection. The incidence of this disease in dogs is reported to be approximately 0.06-6.6% (Sisson and Thomas, 1984). A recent report from western United States showed an incidence of IE 0.9% (MacDonald et al., 2004). In the past, *Staphylococcus aureus, Streptococcus spp., Escherichia coli, Pseudomanas aeruginasa, Erysipelothrix rhusiopathiae*, and *Coryneabacterium* spp. were the most common bacteria associated with IE (Kittleson 1998; Peddle and Sleeper, 2007).

Recently, *Bartonella* spp. have been recognized as a cause of IE in dogs (MacDonald et al., 2004; Pesavento et al., 2005). *Bartonella vinsonii var. berkhoffi* was first described as a cause of canine IE in 1995 (Breitschwerdt et al., 1995). Subsequently, IE has been associated with *B.rochalimae* (Henn et al., 2009), *B. clarridgeiae* (Chomel et al., 2001), *B. quintata* (Kelly et al., 2006), *B.hensalae* (Fenimore et al., 2011), *B.koehlerae* (Ohad et al., 2010) and *B.washoensis* (Chomel et al., 2003). In addition, 28% of dogs affected by IE in western United States had antibody titers against *Bartonella* spp. including *B. vinsonii berkhoffii*, *B. clarridgeiae*, and *B. clarridgeiae-like* (MacDonald et al., 2004).

Seroreactivity to *Bartonella spp.* has been found in 38% of stray dogs (Suksawat et al., 2001) in Thailand. The species found were *B. henselae*, *B. clarridgeiae* and *B. vinsonii supsp. Berkhoffii* (Breitschwerdt et al., 1995; Henn et al., 2001). Dogs in Thailand were also found to be frequently bacteremic with rodent Bartonella species (Kosoy et al., 2010). To our knowledge, the prevalence of IE from any cause is unknown in dogs in Thailand and because of the high seroprevalence of *Bartonella* spp., we hypothesized that *Bartonella* spp. IE occur in dogs in Thailand. The objectives of the study were firstly to determine the prevalence of IE in dogs in Bangkok, Thailand based on necropsy descriptions of the lesions. The second objective was to determine whether bacterial DNA could be amplified from formalin fixed paraffin embedded cardiac tissues using a polymerase chain reaction (PCR) for *Bartonella* spp. DNA and fluorescence *in situ* hybridization (FISH) assay for eubacterial and *Bartonella* spp. DNA.

Materials and Methods

Study population: Necropsy reports of the Department of Veterinary Pathology, Faculty of Veterinary Science, Chulalongkorn University were evaluated for diagnosis of IE from January 1999 to December 2009. Presence of vegetation on any valve leaflets as well as bacteria and inflammatory cells within vegetative leaflets or endocardium were used as the criteria for the diagnosis of IE (Fig 1). All Hematoxylin and Eosin (H&E) stained slides of dogs reported with IE were retrieved and reviewed under light microscope by the same pathologist. Dogs that had lesions in leaflets and endocardium were diagnosed with valvular endocarditis and mural tissue endocarditis, respectively. All dogs affected with IE must have bacteria and/or inflammatory reaction seen in the vegetative lesions or the endocardium on histopathologic sections. Age, gender, breed, clinical findings, and cause of death were recorded for all dogs with IE. The prevalence of IE was calculated by dividing the total cases of IE by the total number of necropsy evaluations performed during the time period X100.

Tissue archives were searched for stored paraffin blocks of all dogs with IE. Paraffin embedded tissue blocks from 11 dogs were shipped to Colorado State University for further evaluation.

Molecular diagnostics: The FISH protocols used were adapted from a previous report (Kornreich et al., 2012). The 4 µm formalin fixed paraffin embedded cardiac tissue sections were deparaffinized by passage of graded alcohol and air-dried. Each slide was initially screened for eubacteria using a combination of an eubacteria probe (EUB-338 Cy3: GCTGCCTCCCGTAGGAGT) and a non-eubacterial control (non-EUB338 6 FAM ACTCCTACGGGAGGCAGC). The slides were subsequently screened using a probe directed against *Bartonella* spp. (ALF 98: GGTAAGGTTCTGCGCGTT) to evaluate for the presence of *Bartonella* spp. DNA

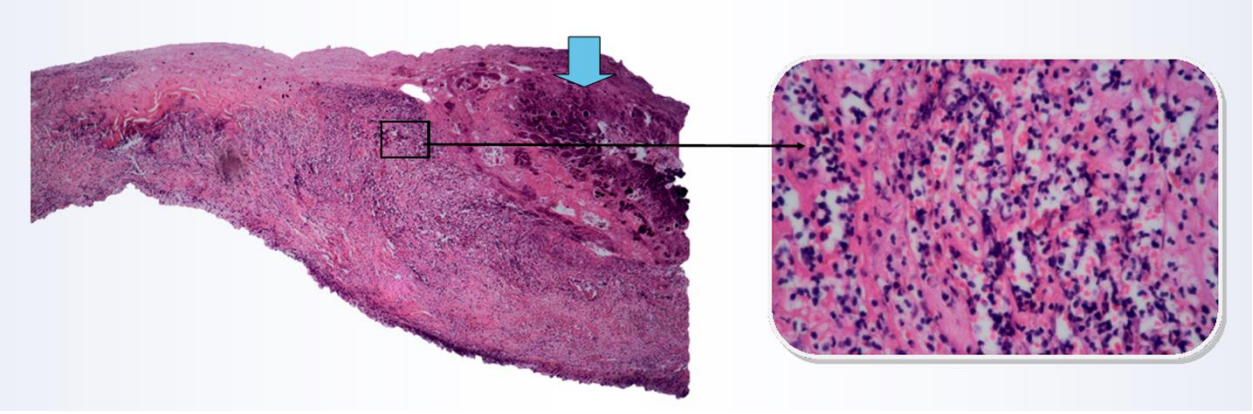

Figure 1 Infectious endocarditis valve with thrombus at the distal part of the valve (arrow) x40 magnification (left). Infiltration of inflammatory cells in the valve stroma x400 magnification (right)

FISH probes (Integrated DNA Technologies, Coralville, IA) were reconstituted with sterile water and diluted to a working concentration of 5 ng/µl with a hybridization buffer (20 mmol/l Tris, 0.9 mol/l NaCl, 0.1 sodium dodecyl sulphate, 40% formamide , pH 7.2). Each section was hybridized with 30-50 µl of hybridization buffer and DNA probe in a humid chamber at 46°C overnight. Washing was done with a washing buffer (20 mmol/l Tris, 0.9 mol/l Na Cl, pH 7.2) at 48°C for 20 min. The slides were rinsed with sterile water, air-dried and counterstained with ProLong Antifade Gold with DAPI (Molecular Probes Inc., Eugene, Oregon, USA). All slides were examined under an Olympus BX51 epifluorescence microscope (Olympus America, Mellville, New York, USA). Images were taken with a DP-70 camera and DP-Manager (Olympus America, Center Valley, Pennsylvania, USA). Sensitivity of these assays for detection and identification of bacteria was 87.5% and specificity was 100% (Kornreich et al., 2012).

A conventional *Bartonella* spp. PCR assay was performed using a previously published protocol (Jensen et al., 2000). Total DNA was extracted from 200 µg of formalin fixed paraffin embedded cardiac tissues from 6 valves and 8 myocardium samples using a commercially available kit (QIAamp DNA Mini Kit, QIAGEN Inc, Valencia, California). The PCR amplifications were performed in 50 µl that contained 10 mM Tris, pH 8.3, 50 mM KCl, 3.5 mM $MgCl_2$, 200 mM each dATP, dCTP, and dGTP, 400 µM dUTP, 1 µM each primer, and 2.5 units Taq polymerase (AmpliTaq Gold DNA polymerase, PE Applied Biosystems, Foster City, Calif). The amplifications were performed in an automated thermal cycler using a protocol including 10 min incubation at 20°C followed by 2 - min denaturation at 95°C then 45 cycles of 1 min at 95°C, 1 min at 60°C, and 30 second at 72°C. PCR amplification products were identified after electrophoresis in 3% agarose gels. Positive and negative samples were included as controls. Sensitivity of this assay in detection of *Bartonella* spp. was 100% of blood samples with 50 to 100 CFU/ml, 85% of blood samples with 30 CFU/ml, and 75% of blood samples with 10 to 20 CFU/ml (Jensen et al., 2000). The sensitivity when used with formalin fixed tissues was unknown.

Results

Study population: Data of 3,545 necropsied dogs were analyzed. Twenty-three were diagnosed with IE giving a prevalence of necropsy cases of 0.65%. Twelve dogs were female and 11 were male. The mean age of dogs was 7.3 ± 3.5 years. Golden retriever was the most common breed (5/23). Other breeds of dogs were 2 German shepherds, 2 Cocker spaniels, 5 mixed breed dogs and one of each Boxer, Collie, Doberman pinscher, Rottweiller, Thai, Mastiff, Siberian, Jack Russell terrier and Poodle.

Clinical findings included heart murmur (26.1%), fever (26.1%), dyspnea (26.1%), vomit (13.0%), anorexia (13.0%), depression (8.7%), diarrhea (8.7%), icterus (4.3%), pale (4.3%), cyanosis (4.3%), cough (4.3%), and exercise intolerance (4.3%). Based on necropsy findings, lesions were noted in the mitral valve alone (30.4%), aortic valve alone (8.7%), both mitral and aortic valves (17.4%), the mural tissue endocarditis alone (26.1%), and the mural tissue endocarditis concurrent with mitral valve endocarditis (17.4%). Of the tissues evaluated by histopathology, bacteria were noted in 17 of 23 cases (73.9%). None of the tissues had been cultured. Suppurative inflammation of other organs was identified by histopathologically and included pneumonia (3.5%), myocarditis (26.1%), lymphadenitis (21.7%), nephritis (17.4%), endometritis (13.0%), enteritis (8.7%), cellulitis (8.7%), hepatitis (8.7%), myositis (4.3%), splenitis (4.3%), peritonitis (4.3%), pancreatitis (4.3%), pleuritis (4.3%), meninigoencephalitis (4.3%), and arthritis (4.3%). Five dogs had endocarditis without other organ inflammation. Most of the dogs died from suspected septicemia (78.3%) thought to result from several causes including cutaneous open wound, gastrointestinal rupture, suppurative pneumonia, urogenital infection, and cancer with secondary bacterial infection. Emboli were seen in 3 dogs (13.0%), causing pulmonary thromboembolism in 2 dogs (8.7%) and myocardial infarction in one dog. Twenty of the 23 (86.9%) dogs had ante-mortem antimicrobial therapy. Ten of the 23 dogs (43.5%) were treated with anti-inflammatory drugs including aspirin tolfenamic acid and caprofen.

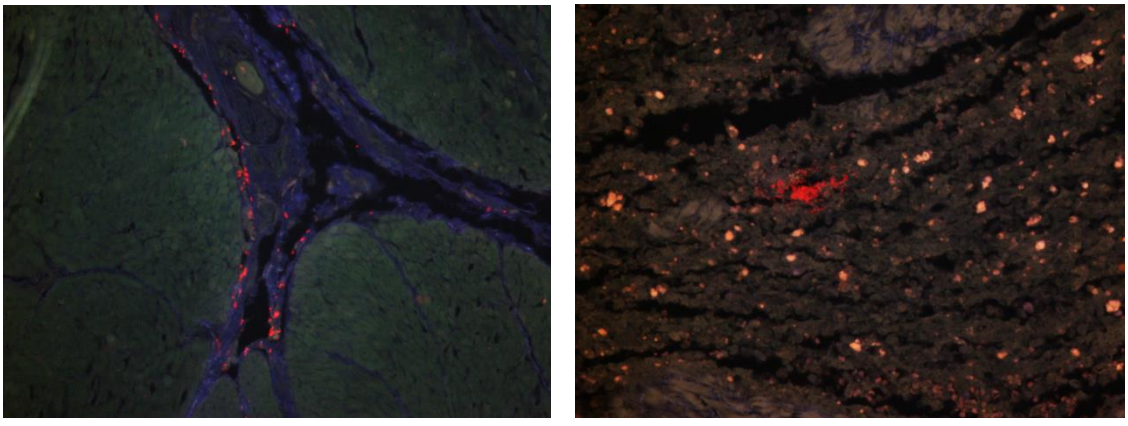

Figure 2 FISH results for paraffin blocks of 2 dogs positive for eubacteria; myocardium (left) and heart valve (right). Eubacteria was hybridized with the EUB-338-Cy3 probe (red). Non-eubacteria was hybridized with the non-EUB338-6 FAM probe (green). DAPI staining for unspecific DNA was revealed as background fluorescence (blue).

Molecular assays: Paraffin blocks from 11 dogs were available for further evaluation. Bacteria were present based on review of H&E stained sections of 7 blocks. The positive and negative controls in all assays performed as expected. Eubacterial DNA was detected by FISH in 2 of 11 dogs (Fig 2). *Bartonella* spp. DNA was not detected by PCR assay or FISH in any tissue.

Discussion

The results of the present study found that the prevalence of canine IE was low in Bangkok, Thailand. These findings are similar to a previously reported prevalence study of dogs from western United States (MacDonald et al., 2004). Almost equal number of males and females were affected in the present study. This is in contrast to previous studies which mostly reported a male: female ratio of 2:1 (Wall et al., 2002; Sykes et al., 2006). The majority of cases were middle-aged, large breeds of dogs. The most affected breed was the Golden retriever. This finding is in agreement with previous studies which found German shepherd, Boxer, Golden retriever, and Labrador retriever as over represented breeds (Wall et al., 2002; Sykes et al., 2006).

Most of the dogs with endocarditis present a combination of non-specific signs including depression, weakness, lethargy, weight loss, anorexia, or fever. These clinical signs are consistent with the findings in this study. The present study demonstrated that 26.1% of the dogs had a heart murmur which is in agreement with a previous study (33%) (Peddle et al., 2009). Thus, auscultation of heart murmur is not a sensitive way to diagnose endocarditis in dogs. This may relate in part to the presence of lesions only on the mural endocardium without involvement of valve leaflets. In this study, only 26.1% of dogs had mural endocarditis alone. Due to the absence of heart murmur and the lack of echocardiographic evidence of disease, mural endocarditis is rarely reported in humans and dogs (Kearney et al., 2004; Miller et al., 2004).

The detection of IE lesions most frequently on the mitral valves is similar to previous studies (Sykes et al., 2006; Peddle et al., 2009). The mitral valves are more affected presumably because these valves have higher risk to be damaged secondarily to an encounter with higher resting pressures for longer length of time (Lepeschkin et al., 1952). The aortic valve was the second most commonly affected valve. Fever was present in only 26.1% of the dogs in the present study indicating that dogs affected with endocarditis may present normal body temperature. Fever may sometimes be obscured by concurrent use of anti-inflammatory drugs (Sykes et al., 2006; Peddle et al., 2009). Almost half of dogs (43.5%) with endocarditis in the present study were treated with anti-inflammatory drugs. These results support those of Peddle et al. (2009), who suggested that a lack of fever was not a good criterion to rule out IE because only half (56%) of the dogs with endocarditis had fever. Based on the results of this study, IE is difficult to diagnose clinically. Only clinical signs and findings from physical examination fail to provide a definitive diagnosis. Although echocardiographic examination can be non-invasively used to reveal lesions within the heart, it has limitations to identify mural lesions and to distinguish between endocarditis and endocardiosis (i.e. degenerative valves) (Boon, 2011). To date, the standardized criterion for assessing patients with IE in veterinary medicine has not been established. Thus, the definitive diagnosis is mostly based on clinical signs including new murmur heart sound and/or fever, laboratory data including positive blood culture results and echocardiographic evidence of endocardial involvement.

In this dog population, suppurative inflammation was seen in several organs including the lungs, lymph nodes, kidneys, uterus, intestines, livers, spleen, muscles, pancreas, peritoneum, pleura, joints and brain. Only 21.7% of the dogs had endocarditis without other organ inflammation. In contrast to a previous report which found 11.84% of dogs with endocarditis to concurrently have arthritis, arthritis was only detected in 4.4% of the IE dogs described here (Peddle et al., 2009). The arthritis associated with IE is thought to occur secondary to deposition of immune complex within the joint, sepsis or emboli (Kittleson, 1998). The dog with arthritis in this study was affected by septic emboli in the joints. Septic emboli are commonly reported in dogs affected with endocarditis and can result in several non-specific clinical signs of disease such as seizure from emboli in brain, diarrhea from intestinal ischemia or sudden death from myocardial infarction (Miller et al., 2004). Emboli were observed in 13.0% in the dogs in the present study and all were detected in the cardiopulmonary system. In another study, the common sites were lungs, kidneys and distal portion of the aorta (Peddle et al., 2009).

The majority of dogs in this study (78.3%) died secondary to sepsis resulting from open wounds, gastrointestinal rupture or diseases with secondary bacterial infection. The remaining dogs presented without septicemia and had no known underlying cause of bacteremia. In most studies, the organisms most commonly associated with IE included *Staphylococcus aureus, Escherichia coli, Pseudomanas aeruginasa* and *Erysipelothrix rhusiopathiae* (Kittleson, 1998). Common associations include *S. aureus* from pyoderma, *E. coli* from the gastrointestinal or urinary tracts, *P. aeruginasa* from chronic wound infections, and *E. rhusiopathiae* from the oral cavity (Calvert and Wall, 2006).

Multiple *Bartonella* spp., including *B. vinsonii var. berkhoffii, B.rochalimae* (Henn et al., 2009), *B. clarridgeiae* (Chomel et al., 2001), *B. quintata* (Kelly et al., 2006), *B.hensalae* (Fenimore et al., 2011), and *B.koehlerae,* have been grown or amplified from dogs with IE (Breitschwerdt et al., 1995; Henn et al., 2009; Chomel et al., 2001; Kelly et al., 2006; Fenimore et al., 2011). These agents are generally vector borne but may also be transmitted by direct contact like bites or scratches. For example, *B. henselae* DNA has been amplified from dog saliva (Duncan et al., 2007a). DNA of several *Bartonella* spp. has been amplified from *Ctenocephalides felis* and *Pulex* spp. collected from dogs (Yore et al., 2012). Ticks are also suspected as vectors. Fleas and ticks are common on dogs in Thailand and *Bartonella* spp. antibodies have been detected in stray dogs, thus, exposure to *Bartonella* spp. in the dogs described herein

would be expected (Nithikathkul et al., 2005; Suksawat et al., 2001).

The present study used previously described FISH protocols to attempt to identify eubacterial or *Bartonella* spp. DNA in the formalin fixed tissues contained in archived blocks from 11 dogs (Kornreich et al., 2012). When compared to H&E staining, one study reported 87.5% sensitivity and 100% specificity of FISH for detection of bacteria in archival heart valve sections (Kornreich et al., 2012). The advantage of FISH over H&E staining and microscopic examination of tissues is the ability to identify the species of causative bacteria. Moreover, FISH can be used to localize bacteria within histological sections while conventional PCR techniques cannot (Moter and Göbel, 2000).

While 7 of the 11 dogs had bacteria visualized on H&E stained sections, eubacteria were only detected in 2 dogs by FISH. The positive tissues were endocardium for one case and valve tissue for the other case. Of these 2 cases, bacteria were also seen histopathologically. The failure to document eubacterial DNA by FISH in the other 5 histopathologically positive samples suggest that the bacteria visualized were not actually bacteria or that the FISH was falsely negative. None of the cases were evaluated by blood or tissue culture and so further information to aid in determining which possibility is true is not available. All 11 dogs were treated with antibiotics before death which may have affected the results of both assays. In future studies, it would be optimal to collect fresh tissues for culture as well as molecular assays.

None of the dogs in the present study was positive for *Bartonella* spp. by FISH or conventional PCR performed on the archived formalin fixed tissues. *Bartonella* spp. DNA can be amplified from formalin fixed cardiac tissues from dogs and the conventional *Bartonella* spp. assay used is relatively sensitive (Fenimore et al., 2011; Jensen et al, 2000). These results suggest that *Bartonella* spp. were not the cause of IE in this population of dogs. Another recent report from mid-Atlantic United States also failed to detect *Bartonella* spp. in any archival valve sections from dogs with suspected IE (Kornreich et al., 2012). These findings may indicate that biological behavior of *Bartonella* spp. strains may vary regionally as has been documented with *B. henselae* infection in cats. While most *B. henselae* strains in cats induce only subclinical infections, one strain studied in experimentally exposed cats induced cardiac disease in 2 of 6 cats (Bradbury and Lappin, 2010). In one study, *Bartonella* spp. was mostly found at aortic valves (Pesavento et al., 2005), however, most of the dogs in this study had mitral valve endocarditis which may have also influenced the results. A larger study that combines more sensitive techniques should be performed using blood and fresh tissues. The use of pre-enrichment liquid culture followed by PCR has been shown to be more sensitive than other methods for detection of *Bartonella* spp. infections in dogs (Duncan et al., 2007b; Bai et al., 2010).

Acknowledgements

This study was supported by the Grants for Development of New Faculty Staff, Ratchadaphiseksomphot Endowment Fund. The authors wish to thank the Department of Veterinary Pathology, Chulalongkorn University, Thailand for data and sample collection, the Center for Companion Animal Studies, Department of Clinical Sciences, Colorado State University for performing PCR and FISH and the Department of Clinical Sciences, College of Veterinary Medicine, Cornell University, USA for technical and laboratory support.

References

Bai Y, Kosoy MY, Boonmar S, Sawatwong P, Somboon S and Leonard FP 2010. Enrichment culture and molecular identification of diverse *Bartonella* species in stray dogs. Vet Microbiol. 146: 314-319.

Boon AJ. 2011. Acquired valvular disease. In: Veterinary echocardiography. 2nd ed. Ames: Willey Blackwell. 267-358.

Breitschwerdt EB, Dorsey LK, Malarkey DE, Keene B, Hadfield TL and Wilson K 1995. Endocarditis in a dog due to infection with a novel *Bartonella* subspecies. J Clin Microbiol. 33: 154-160.

Bradbury CA and Lappin MR 2010. Evaluation of topical application of 10% imidoclopid-1 % moxidectin to prevent Bartonella henselae transmission from cat fleas. J Am Vet Med Assoc. 15: 869-873.

Calvert CA and Wall M 2006. Cardiovascular infections. In: Infectious diseases of the Dog and Cat. 3rd ed. Green CE (ed.) St. Louis, Elsevier Saunders. 841-854.

Chomel BB, MacDonald KA, Karsten RW, Chang CC, Wey AC, Foley JE, Thomas WP and Kittleson MD 2001. Aortic valve endocarditis in a dog due to *Bartonella clarridgeiae*. J Clin Microbiol. 39: 3548-3554.

Chomel BB, Wey AC and Kasten RW 2003. Isolation of *Bartonella washoensis* from a dog with mitral valve endocarditis. J Clin Microbiol 41: 5327-5332.

Duncan AW, Maggi RG and Breitschwerdt EB 2007a. Bartonella DNA in dog saliva. Emerg Infect Dis. 13: 1948-1950.

Duncan AW, Maggi RG and Breitchwerdt EB 2007b. A combined approach for the enhanced detection and isolation of Bartonella species in dog blood samples: pre-enrichment liquid culture followed by PCR and subculture onto agar plates. J Microbiol Methods 69: 273-281.

Fenimore A, Varanat R, Maggi P, Schultheiss P, Breitschwerdt E and Lappin MR 2011. *Bartonella* spp. DNA in cardiac tissues from dogs in Colorado and Wyoming. J Vet Intern Med. 25: 613-616.

Henn JB, Gabriel MW, Karsten RW, Brown RN, Koehler JE, MacDonald KA, Kittleson MD, Thomas WP and Chomel BB 2009 . Infective endocarditis in a dog and the phylogenetic relationship of the associated "*Bartonella rochalimae*" strain with isolates from dogs, gray foxes, and a human. J Clin Microbiol 47: 787-790.

Jensen WA, Fall M, Rooney J, Kordick DL and Breitschiwerdt EB 2000. Rapid identification and differentiation of *Bartonella* species using a single step PCR assay. J Clin Microbiol. 38: 1717-1722.

Kearney RA, Eisen HJ and Wolf JE 1994. Nonvalvular infections of the cardiovascular system. Ann Intern Med. 121: 219-230.

Kelly P, Rolain JM, Maggi R, Sontakke S, Keene B, Hunter S, Lepidi H, Breitschwerdt KT and Breitschwerdt EB 2006. *Bartonella quintana* endocarditis in dogs. Emerg Infect Dis. 12: 1869-1872.

Kittleson MD 1998. Infective endocarditis. In: Small animal cardiovascular medicine. Kittleson MD and Kiele RD (ed.) St. Louis, CV Mosby Inc 402-412.

Kornreich BG, Craven M, McDonough SP, Nydam DV, Scorza V, Assarasakorn S, Lappin M and Simpson KW 2012. Fluorescence In situ hybridization for the identification of bacterial species in archival heart valve sections of canine bacterial endocarditis. J Comp Pathol. 146: 298-307.

Kosoy M, Bai Y, Sheff K, Morway C, Baggett H, Maloney SA, Boonmar S, Bhengsri S, Dowell SF, Sitdhirasdr A, Lerdthusnee K, Richardson J and Persuki LF 2010. Identification of *Bartonella* infection in febrile human patients from Thailand and their potential animal reservoirs. Am J Trop Med Hyg. 82: 1140-1145.

Lepeschkin E 1952. On the relation between the site of valvular involvement in endocarditis and the blood pressure resting on the valve. Am J Med Sci. 224: 318-322.

MacDonald KA, Chomel BB, Kittleson MD, Karsten RW, Thomas WP and Pesavento P 2004. A prospective study of canine bacterial endocarditis in northern California (1999-2001): emergence of *Bartonella* as a prevalent etiologic agent. J Vet Intern Med. 18: 56-64.

Miller MW, Fox PR and Saunders AB 2004. Pathologic and clinical features of infective endocarditis. J Vet Cardiol. 6: 35-43.

Moter A and Göbel UB 2000. Fluorescence in situ hybridization (FISH) for direct visualization of microorganisms. J Microbiol Methods. 41: 85-112.

Nithikathkul C, Polseela R, Iamsa-ard J, Wongsawad C and Jittapalapong S 2005. A study of ectoparasites of *Canis lupus familiaris* in mueang district, Khon Kaen, Thailand. Southeast Asian J Trop Med Public Health. 36 (suppl 4): 149-151.

Ohad DG, Morick D, Avidor, B and Herrus S 2010. Molecular detection of *Bartonella henselae* and *Bartonella koehlerae* from aortic valves of Boxer dogs with infective endocarditis. Vet Microbiol. 141: 182-185.

Peddle G and Sleeper MM 2007. Canine bacterial endocarditis: a review. J Am Anim Hosp Assoc. 43: 258-263.

Peddle GD, Drobatz KJ, Harvey CE, Adams S and Sleeper MM 2009. Association of periodontal disease, oral procedures, and other clinical findings with bacterial endocarditis in dogs. J Am Vet Med Assoc. 1: 100-107.

Pesavento PA, Chomel BB, Kasten RW, McDonald KA and Mohr FC 2005. Pathology of *Bartonella* endocarditis in six dogs. Vet Pathol. 42: 370-373.

Sisson D and Thomas WP 1984. Endocarditis of the aortic valve in the dog. J Am Vet Med Assoc. 184: 570-577.

Suksawat J, Wuejie Y, Hancock S, Hegarty BC, Nilkumhang P and Breitschwerdt EB 2001. Serologic and molecular evidence of co-infection with multiple vector borne pathogen in dogs in Thailand. J Vet Intern Med. 15: 453-462.

Sykes JE, Kittleson MD, Chomel BB, MacDonald KA and Pesavento PA 2006. Clinicopathologic findings and outcome in dogs with infective endocarditis: 71 cases (1992-2005). J Am Vet Med Assoc. 228: 1735-1747.

Wall M, Calvert CA and Green CE 2002. Infective endocarditis in dogs. Compend Contin Educ Vet. 24: 614-625.

Yore K, DiGangi B, Brewer M and Lappin MR 2012. Determination of the fleas species infesting dogs in Florida and *Bartonella* spp. prevalence rates. 2nd Biennial Symposium of the International Society for Companion Animal Infectious Diseases (ISCAID). November 14-17, 2012, San Francisco, CA, USA.

บทคัดย่อ

ความชุกของโรคเยื่อบุหัวใจอักเสบติดเชื้อในสุนัขและความเป็นไปได้ของการติดเชื้อบาโทเนลลา ในเขตกรุงเทพมหานคร ประเทศไทย

สิริลักษณ์ ดิษเสถียร สุรเชษฐพงษ์[1*] สุกัลยา อัศรัสกร[1] อนุเทพ รังสีพิพัฒน์[2] เอ วาเลอเรีย สคอร์สา[3]

เมลลิสสา เอ็ม บรูเวอร์[3] เคนเนธ ดับเบิ้ลยู ซิมสัน[4] ไมเคิล อาร์ แลปปิน[3]

โรคเยื่อบุหัวใจอักเสบติดเชื้อเป็นโรคของลิ้นหัวใจหรือผนังเยื่อบุหัวใจชั้นใน เชื้อบาโทเนลลาเป็นสาเหตุหนึ่งของการเกิดโรคเยื่อบุหัวใจอักเสบติดเชื้อ วัตถุประสงค์ของงานวิจัยนี้เพื่อศึกษาความชุกของโรคเยื่อบุหัวใจอักเสบติดเชื้อในสุนัข ในเขตกรุงเทพมหานคร ประเทศไทย โดยเน้นความสัมพันธ์กับการติดเชื้อบาโทเนลลา ทำการศึกษาโดยทบทวนรายงานผลชันสูตรซากสุนัขจำนวน 3545 ตัว ระหว่างเดือนมกราคม 2542 ถึงเดือนธันวาคม 2552 และศึกษาการปรากฏของดีเอ็นเอของยูแบคทีเรียและเชื้อบาโทเนลลา จากชิ้นเนื้อของกล้ามเนื้อหัวใจจำนวน 11 ชิ้น โดยวิธีฟลูโอเรสเซนต์ อินไซตูไฮบริดไดเซชั่น และปฏิกิริยาลูกโซ่โพลิเมอเรส พบความชุกของโรคโรคเยื่อบุหัวใจอักเสบติดเชื้อจากการติดเชื้อในสุนัขร้อยละ 0.65 พบยูแบคทีเรียดีเอ็นเอจากการตรวจด้วย ฟลูโอเรสเซนต์ อินไซตู ไฮบริดไดเซชั่น ในกล้ามเนื้อหัวใจ 2 จาก 11 ตัวอย่าง ไม่พบผลบวกต่อเชื้อบาโทเนลลาด้วยการตรวจทั้ง 2 วิธี โดยสรุปความชุกของโรคเยื่อบุหัวใจอักเสบติดเชื้อในสุนัขค่อนข้างต่ำในประชากรสุนัขที่ทำการศึกษา และไม่พบการปรากฏของดีเอ็นเอของเชื้อบาโทเนลลาจากวิธีการที่ทำการศึกษาในครั้งนี้

คำสำคัญ: เชื้อบาโทเนลลา สุนัข ลิ้นหัวใจอักเสบ หัวใจ ประเทศไทย ลิ้นหัวใจ

[1]ภาควิชาอายุรศาสตร์ คณะสัตวแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย กรุงเทพฯ 10330

[2]ภาควิชาพยาธิวิทยา คณะสัตวแพทยศาสตร์ จุฬาลงกรณ์มหาวิทยาลัย กรุงเทพฯ 10330

[3]ภาควิชาวิทยาศาสตร์ทางคลินิก มหาวิทยาลัยแห่งรัฐโคโลราโด ฟอร์ท คอลลิน โคโลราโด ประเทศสหรัฐอเมริกา 80523

[4]ภาควิชาวิทยาศาสตร์ทางคลินิก วิทยาลัยสัตวแพทย์ มหาวิทยาลัยคอร์เนล อิธาคา นิวยอร์ก ประเทศสหรัฐอเมริกา 14583

****ผู้รับผิดชอบบทความ E-mail:** sirilakd27@gmail.com*